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CIGARETTE SMOKING AND LOW BACK PAIN

by

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Bachelor of Science in Physical Therapy
University of North Dakota, 1984

An Independent Study

Submitted to the Graduate Faculty of the

Department of Physical Therapy

School of Medicine

University of North Dakota

in partial fulfillment of the requirements

for the degree of

Master of Physical Therapy

Grand Forks, North Dakota
May
1993

This Independent Study, submitted by Cindy G. Broden in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the chairperson of Physical Therapy under whom the work has been done and is hereby approved.

(Chairman, Physical Therapy)

PERMISSION

Title Cigarette Smoking and Low Back Pain

Department Physical Therapy

Degree Master of Physical Therapy

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ABSTRACT

The purpose of this independent study is to define presently existing theories linking cigarette smoking and low back pain. It also provides a detailed description of nicotine and its physiological and psychological effects.

There are four theories outlined in this study which could serve as explanations for the correlation of cigarette smoking and low back pain. The first is the chronic cough associated with smoking. This cough increases mechanical stress including higher disc pressures and poor union and healing following spinal fusion surgery. The second theory deals with the vascular changes caused by nicotine which decreases vertebral blood flow and solute transport. Thirdly, smoking accelerates osteoporosis. The last theory links cigarette smoking and low back pain with similar psychological and physiological traits.

There is enough evidence to show a strong correlation between cigarette smoking and low back pain, however no conclusive evidence exists to demonstrate cigarette smoking as a causative factor of low back pain. The information is strong enough to alert the healthcare provider to not overlook cigarette smoking when treating a patient with low back pain.

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CHAPTER I

INTRODUCTION

Cigarette smoking is generally considered to be hazardous to your health. Smoking is thought to increase risks of coronary heart and artery disease, peripheral vascular disease, chronic obstructive lung disease, bronchitis, emphysema, and cancer of the lungs, oral cavity, larynx, pharynx, esophagus, and bladder.[1-4] There are also several sources which link cigarette smoking to low back pain.[5-12]

In addition to cigarette smoking, many other risk factors have been identified for low back pain. These include truck driving, extensive automobile driving, prolonged sitting, emotional stress, vibrational exposure, lifting, bending, poor posture, psychological factors such as depression and anxiety, decreased physical fitness, and repetitive activities.[6,7,9,10,13-15] Obesity and age have also been considered risk factors.[8]

When looking at the relationship of cigarette smoking and low back pain, several explanatory theories can be found. Biering-Sorenson and Thomsen[10] postulated that smoking produces a chronic cough which in turn gives rise to increased mechanical stress including higher disc pressures.

Another study that stated cigarette smoking dramatically decreased the chances for successful disc surgery felt that a chronic cough could cause poor union and poor healing, thereby increasing pain.[12]

Cigarette smoking has also been thought to reduce vertebral blood flow.[7,8] Smoking activates the sympathetic nervous system, resulting in increased heart rate and blood pressure, increased cardiac stroke volume and output, and increased coronary blood flow.[16,17] It also causes peripheral vasoconstriction.[16] Since the disc depends on diffusion through vertebral end-plates, smoking may adversely affect discal metabolism, increasing susceptibility to mechanical injury.[7,8,10,12]

Another theory deals with osteoporosis. Women smokers are shown to have significantly lower spine bone mineral density and a tendency for lower bone mineral density at other sites.[9,18] Consequently, there is a possible correlation between smoking and vertebral osteoporosis. Microfractures of the trabeculae in the lumber vertebrae caused by osteoporosis might be responsible for some low back pain.[9,10]

Cigarette smoking and low back pain have also been linked by psychological and social traits rather than mechanical and physiological causes.[8,9,19-21] Chronic smokers may have a psychological profile that both relates to the smoking habit itself and also places them at greater

risk of symptomatic low back pain.[8] The smoking habit is found to be more prevalent in lower socioeconomic groups for whom job demand, life stress, income, and other health habits may enhance the risk of back pain.[8] Stress, worry, and tension are associated with both low back pain and cigarette smoking.[9]

Information regarding these theories is very scant in current literature. The question remains as to whether cigarette smoking is actually a causative factor, a risk indicator or a result of low back pain. Jamison[5] stated that persons who reported having pain in general showed a higher rate of smoking. As pain severity increased, there was a tendency to exercise less and smoke more. Many persons report they smoke to relieve pain and anxiety.[22-27] This would seem to imply that low back pain caused increased smoking, therefore smoking may not be a causative factor. There is no conclusive evidence connecting cigarette smoking and low back pain, however, there is enough information in current literature to show a strong correlation.

CHAPTER II

METHODOLOGY

This study will be a review of currently available literature pertaining to nicotine and its numerous effects on the human body. Areas included in this literature search are the psychology and pharmacology of nicotine, risk factors associated with low back pain, personality traits related to low back pain as well as cigarette smokers and relationships between cigarette smoking and low back pain.

The aim of this study is to compile information from this literature search regarding the presently existing theories supporting the hypothesis that cigarette smoking may be a significant risk factor for low back pain.

CHAPTER III

NICOTINE

History of Cigarette Smoking

The tobacco plant, nicotiana tabacum, is surely the most loved and hated member of the vegetable kingdom.[1] The tobacco plant is indigenous to the Americas. Natives of the Americas had smoked and chewed these herbs for a variety of narcotic, stimulant, medicinal, social, and religious reasons for a long time prior to the arrival of European explorers.[3] Columbus discovered tobacco in his discovery of America in 1492. It was subsequently brought to Europe by future explorers in the sixteenth century. Portuguese and Spanish sailors were introducing tobacco to the far corners by the end of the sixteenth century.[3]

Tobacco usage was opposed from the outset and anti-smoking measures were taken from the earliest times. Murad the Cruel of Turkey (1623-1640) had smokers beheaded, hung, and quartered.[3] In the Russia of the first Czar, smokers were flogged and exiled to Siberia.[3] In Japan in 1616, the penalties for smoking included imprisonment and the confiscation of property.[3] Surprisingly, severe and immediate punishment proved no more effective in halting

smoking than current fears of long-term health consequences.[3] Nearly 30 percent of adult Americans smoke despite, in most cases, a desire to quit and despite common knowledge of the health hazards.[16]

Description of Nicotine

Cigarette smoke is a complex mixture of gases and tar particles containing scores of organic compounds, but there is little doubt that nicotine is the pharmacological agent of prime importance.[1] In the gas phase, the major constituents are nitrogen, oxygen, and carbon dioxide.[3] In the residual gases, carbon monoxide is present in significant concentration, together with smaller amounts of other gases including isoprene, acetaldehyde, acetone, and hydrogen cyanide.[3] Smoking doses of nicotine are known to exert profound effects on the central nervous system and behavioral actions, the cardiovascular system, endocrine and metabolic action, and neuromuscular action.[16]

Nicotine is a tertiary amine composed of a pyridine and a pyrrolidine ring.[16] It is colorless, volatile, and strongly alkaline in reaction.[28] On exposure to air, it turns brown and acquires the odor typical of tobacco. The free base is present in the smoke suspended on minute droplets of tar, which are small enough to reach the small airways and lung alveoli.[16,28] At a physiologic pH, about

thirty-one percent is un-ionized and can readily cross cell membranes.[16]

Nicotine is pharmacologically active in the form of the nicotinium ion.[3] The structural basis for the pharmacological activity of the nicotinium ion would appear to be its resemblance to acetylcholine in terms of the spacing of positive and negative charges. Acetylcholine (ACh) is a chemical neurotransmitter which conveys information from one nerve cell to another. The ability of nicotine to combine with ACh receptors means that it can exert actions like ACh at all synapses where nicotinic ACh receptors are present.[1] These receptors are found at the spinal cord, autonomic ganglia, the adrenal medulla, neuromuscular junctions, and the brain.[16] As a consequence of the stimulation of nicotinic receptors, possibly located on presynaptic sites, short-term exposure to nicotine results in the activation of several central nervous system neurohumoral pathways, leading to the release of acetylcholine, norepinephrine, dopamine, serotonin, vasopressin, growth hormone, and ACTH.[16]

Nicotine readily crosses the blood-brain barrier and is distributed throughout the brain. Absorption through the lungs is very nearly as efficient as an intravenous injection. More than ninety percent of the nicotine in inhaled cigarette smoke is absorbed by the lungs.[3] Following inhalation of cigarette smoke, nicotine will reach

the brain in about eight seconds and the extremities in approximately twenty seconds.[1,3]

The effects of nicotine are complex and sometimes confusing as nicotine has biphasic effects due to its ability to interact with acetylcholine receptors.[16,28] In low doses, nicotine has ganglionic stimulation, but in high doses produces ganglion blockade.[16] In general, small doses produce a predominantly stimulant effect at synapses, and larger doses produce mainly a depressant effect.[1,3,16,22,28,29]

Cigarette smoking also activates the sympathetic nervous system. This produces cardiovascular effects of increased heart rate, increased blood pressure, increased cardiac stroke volume and output, and increased coronary blood flow.[1,3,16,17,25,28-30] This is accompanied by cutaneous vasoconstriction, decreased skin temperature, systemic venoconstriction, and increased muscle blood flow.[1,3,16,23,28,29,31] There is also an increase in norepinephrine and epinephrine as well as a release of vasopressin.[16] The effects of norepinephrine and epinephrine are similar to those brought about by stimulation of the sympathetic nervous system.[32]

Physiological Effects of Nicotine

There are numerous physiological effects of nicotine. Cigarette smoking may contribute to atherosclerotic vascular

disease through its effect on hemostasis.[16] Blood coagulates more easily in smokers than in nonsmokers, fibrinogen levels are higher, and platelets have been reported in some studies to be more reactive.[16] Platelet survival is shortened in smokers as compared with nonsmokers.[16] The role of nicotine in causing platelet hyperaggregability remains to be established.

In women, cigarette smoking is associated with earlier menopause and increased risk of osteoporosis, believed to be associated with lower levels of estrogen in smokers compared to nonsmokers.[9,10,16,18,33-37] Cigarette smoking doubles the risk of osteoporosis.[37] Researchers have found that osteoporosis happens earlier, and continues more rapidly, in smokers than in nonsmokers.[37] Recent evidence suggests that nicotine and other alkaloids in tobacco inhibit the formation of estrogen by inhibiting an aromatase enzyme in granulosa cells and placental tissue.[16] Another study showed tobacco smoke reduced the sensitivity to calcitonin.[36] Calcitonin protects bone tissue against resorptive effects, hence decreased sensitivity to it may offer an explanation for the osteoporogenic effect of smoking, especially because calcitonin resistance can be found in pathologic menopausal osteoporosis.[36]

Nicotine can also cause a muscle contraction due to the response of the nicotinic receptors to released acetylcholine.[28,29] There is an ion flux that can

depolarize the muscle cell, resulting in muscular contraction.[28,29] When the nicotine molecule first hits the receptors, it helps initiate a muscular contraction, but this is followed by prolonged paralysis, possibly due to blocking of subsequent depolarization.[29] With high doses, one may obtain a net effect of prolonged receptor blockade.[29]

One study showed nicotine to cause a dramatic transient reduction in skeletal muscle tone in spastic patients.[3] Nicotine also inhibits phasic stretch reflexes, like the patellar reflex.[3,16,22] This decreased patellar reflex is moderated by a direct stimulant action of nicotine on cholinergic receptors of the Renshaw cells in the spinal cord.[3,16] Studies of the trapezius and forearm extensors showed increased tonic muscle activity with nicotine as well as an increased muscle tremor.[3] It is possible that stimulant effects may occur in one set of muscles and depressant effects in others.

Cigarette smoking is also thought to influence the response to many drugs, causing failure of medical therapy in some cases, or drug toxicity.[2,16] Smoking interacts with medications through its effects on drug metabolism and pharmacodynamics. It accelerates metabolism of many drugs, thereby requiring larger doses.[2,16] Drugs with accelerated metabolism are antipyrine, caffeine, desmethyldiazepam,

imipramine, lidocaine, oxazepam, pentazocine, phenacetin, propranolol, and theophylline.[16]

Cigarette smoking can simulate chronic inflammation.[38] It has been used to investigate both the involvement of phagocyte-derived reactive oxidants in the pathogenesis of pulmonary dysfunction and carcinogenesis, as well as the possible role of nutritional antioxidants in the prevention of oxidant-mediated tissue damage.[38] Reactive oxidants generated by activated phagocytes appear to be primary mediators of spirometric abnormalities in cigarette smoking.[38] Cigarette smoke is also associated with decreased plasma levels of ascorbate and beta-carotene, which indicates that the smoking-related chronic inflammatory response leads to an imbalance of oxidant/antioxidant homeostasis and possible predisposition to oxidant-inflicted tissue damage and disease.[38]

As mentioned earlier, another effect of nicotine is cardiovascular, due to the activation of the sympathetic nervous system. The increased coronary blood flow and decrease in peripheral blood flow play a role in the physiological effects of nicotine in relation to low back pain.

Psychological Effects

Many psychological changes are attributed to cigarette smoking. These changes may be due to psychological as well

as pharmacological reasons.[28] Behavioral effects of nicotine include changes in the rates of conditioned and unconditioned behaviors, a variety of stimulus properties, and possible influences on cognitive function.[16,29] Many smokers report that cigarette smoking improves their mood and they feel more relaxed.[22,25,27,28] Even though smokers report feeling relaxed, cigarette smoking has a physiologically stimulating effect.[24,25] Smokers also use cigarette smoking to combat boredom and fatigue.[25] Many smokers also claim smoking helps them to think more clearly and to concentrate, thereby enhancing performance on tasks like visual surveillance and rapid information processing as well as long-term memory.[22]

Smoking has also been reported to decrease pain as well as anxiety.[29] Several studies have reported increased pain threshold and pain tolerance after smoking.[22,23,26] Other studies have reported no change in pain threshold and tolerance and actually a decrease in deep pain tolerance.[26,39] The function of nicotine remains questionable in regard to pain tolerance as well as anxiety. Some researchers believe that smokers are trying to avoid the withdrawal symptoms of nicotine, rather than reducing anxiety, by smoking in stressful situations.[23,27] Stress has been shown to deplete levels of nicotine in stressed smokers, which may also explain the smokers desire to smoke to reduce anxiety and stress.[27]

Personality Traits

Just as smokers and non-smokers react differently to stressful and painful situations, they also differ to a measurable extent in certain genetically-based characteristics, including personality. Smokers show more prevalence in changing jobs frequently, higher rates of divorce, lower academic achievement, drinking more, rebellion against authority, lower levels of physical activity, and difficulty sleeping.[1,3] Adults from lower socioeconomic classes smoke more than those from higher classes.[2] Smokers also show personality traits of defiance, impulsivity, and danger-seeking, and were relatively high in manifest distress, had childhood experiences that were relatively barren of emotional support, and showed oral preoccupation.[2] Smokers are found to be more extroverted as compared to nonsmokers.[1-3] There is also evidence that smokers may be predisposed to emotional disturbance.[40]

There may well be other uncharted personality dimensions and undiscovered brain mechanisms upon which nicotine may act. Despite these uncertainties and complexities, the literature on smoking and personality justifies two conclusions relevant to smoking motivation. First, there is evidence that the effects of nicotine vary according to the personality and situation of the smoker.[1] Secondly, there are small but statistically reliable personality differences between the population of smokers

and the population of non-smokers.[1] The many effects of nicotine on brain and behavior, and the interaction of these with individual differences, suggest that many smokers, in their own way, are engaged in manipulating their psychological state through the effects of smoking on arousal and reward punishment systems.[1] For example, the reported effects of reduced pain and anxiety and the ability to concentrate and think more clearly are positive reinforcements for smoking. How smokers regulate the effects of nicotine is dependent upon the individual's personality and psychological traits.

CHAPTER IV

THEORIES OF CIGARETTE SMOKING AND LOW BACK PAIN

Chronic Cough

Cigarette smoking has been shown to have numerous effects on the human body, both physiological and psychological. The mechanical effect of chronic cough on intradiscal pressure is one theory as to why cigarette smoking may be a risk factor in low back pain. Coughing and smoking are clearly related.[35] This increased mechanical stress of higher disc pressure induced by a chronic cough could contribute to disc disease and herniation.[6-11,35,41-43]

A significant portion of severe and prolonged low back pain is attributable to the herniated lumbar disc.[44] The protrusion of disc material posteriorly or posteriolaterally results in pressure on the spinal cord, cauda equina, or the spinal nerves giving rise to neurologic symptoms.[32] Bulging or herniation of a portion of the nucleus pulposus through the annulus fibrosus occurs as a result of sudden compressive forces above and below the spinal vertebra which are sufficient to rupture the fibers of the annulus.[32] Coughing can produce this compressive force as coughing is

known to increase intradiscal pressure. Many activities which increase intradiscal pressure, such as forward bending, slumped sitting, sneezing and coughing, have been found to be significant risk indicators for low back pain.[6,7,13,44] Therefore it is possible that a chronic cough from smoking may be a causative factor for low back pain due to increased intradiscal pressure. However, Frymoyer et al.[7] indicated that smoking and coughing were related to low back pain, but that coughing alone was insufficient to account for the observed differences in back pain complaints in subjects who smoked.

McFadden[12] found surgical success of spinal fusions was two times more frequent for nonsmokers than for smokers. He attributes poor union and slow healing or continued injury of the lumbar disc partly to chronic coughing from smoking.[12] An upper respiratory infection or bronchitis is frequently the precipitating factor in the exacerbation of disc pain, with onset commonly associated with coughing or sneezing.[12] An association between low back pain and chest symptoms such as coughing, wheezing, and shortness of breath, has also been suggested.[35] This could stem from the mechanical effects of coughing or even from behavioral patterns created by the experience of back pain.[35]

In reviewing the existing studies, it does seem conceivable that the smokers cough could contribute to low

back pain in the form of disc disease and poor surgical healing.

Vascular Changes

Cigarette smoking is also thought to be a risk factor of low back pain because of its effect on the circulatory system. Cardiovascular effects of nicotine are increased cardiac stroke volume and output, increased coronary blood flow, cutaneous vasoconstriction, decreased skin temperature, systemic venoconstriction, and increased muscle blood flow.[1,3,16,23,25,28-31] Defects in fibrinolysis and association with the atherosclerotic process are also associated with smoking.[35,45-48] The circulatory changes seen with cigarette smoking can have detrimental effects on disc metabolism in several ways.

Nutrition of the vertebral disc depends on diffusion through the vertebral end-plates.[7,8,10,12,49-51] The disposal of metabolic waste products also depends on exchange with the blood vessels outside the disc.[49] Nutritional deficiencies could lead to disc degeneration.[49] In intervertebral discs where the balance between nutrient utilization and supply is precarious, any loss in blood vessel contact or reduction in blood flow at the periphery of the disc could lead to nutritional deficiencies and build-up of waste products.[14,49,51]

The blood supply is located in the outer zone of the intervertebral segment.[50] These blood vessels originate from the vascular network surrounding the spinal column and enter the annulus fibrosus directly.[50] They perforate its lamellae radially and form intralamellar capillary networks, but they do not enter the inner layers of the annulus fibrosus or the nucleus pulposus.[50] From the beginning, nutrition of the central segments of the disc is by diffusion, whereas the cartilaginous end-plates are well vascularized with peripheral central axial blood vessels.[50] If the end-plate becomes partially blocked, there would be danger of build-up of waste products such as carbon dioxide and lactic acid, or of the development of a nutrient deficiency of the central segment of the disc which may lead to disc degeneration.[49]

As smoking increases coronary blood flow and decreases peripheral blood flow, it is thought to decrease vertebral blood flow which adversely affects disc metabolism and makes it susceptible to injury.[7,8,10,12] In one study, the results showed a significant reduction in solute transport after exposure to cigarette smoke.[41,51] Diffusion of sulphate, oxygen, and methylglucose was reduced by thirty to forty percent.[51] This effect was obtained after exposure to smoke for twenty to thirty minutes. A smoking period of three hours reduced the transport efficiency to about fifty percent.[51]

Smoking has also been found to decrease the oxygen tension in discs.[52] The oxygen-carrying capacity of the blood is diminished in chronic smokers as a result of elevated carboxyhemoglobin levels.[46] Carbon monoxide, a main component of cigarette smoke, is irreversibly bond to hemoglobin.[46] Although cells within articular cartilage, and presumably within the disc as well, are able to function anaerobically, very low oxygen tension may adversely affect certain processes such as glycosaminoglycon production.[49]

Battie et al.[53] completed a study of monozygotic twins and detected a possible anatomic cause for the increased risk of back pain associated with smoking. The lumbar spines of twenty sets of twins highly discordant for cigarette smoking were studied by magnetic resonance images. Intervertebral disc degeneration was increased at all lumbar levels by a mean of 18 percent in the smoking twin.[53] The disc metabolism was thought to be affected either by chemical factors or vasopressin resulting in degeneration.[53]

Pseudarthrosis, or surgical nonunion, is more likely to develop in smokers because of inadequate oxygenation of blood flow to the bone graft.[54] Brown et al.[54] found that patients who were habitual smokers generally had lower blood gas levels, thought to be due to increased carbon monoxide absorption and to smoking-produced arterial constriction. The result of the inadequate oxygenation is the formation of fibrous tissue rather than bone.[54] In some cases, at the

time of exploration of the non-union site, there was no evidence of any previous graft material at all.[54] It appeared to have been absorbed completely and replaced by fibrous tissue.[54] The results of the Brown[54] study appear to demonstrate clearly that cigarette smoking is a highly significant factor in the failure-to-fuse syndrome in posterior lateral spinal fusion as fusion failed in approximately 40 percent of the smokers, but in only eight percent of the nonsmokers.[12,54]

The fibrinolytic effects associated with smoking also suggest that smoking could have an etiological effect in disc disease.[35] The fibrinolytic activity has been found to be lower and fibrinogen concentration higher in smokers as compared to nonsmokers.[47,48,55] It has been suggested that the abnormal persistence of a defect in fibrinolytic activity, leading to fibrin deposition and chronic inflammation, may be an important factor in the chronicity of many back pain syndromes.[48,55]

Persistent deposition of fibrin could block the diffusion of oxygen with consequent ischemia of the nerve roots if it occurs around the capillaries.[55] In one study with chronic back pain patients, 50 percent were smokers compared with 18 percent of controls.[48] A defect in fibrinolysis has been associated with low back pain as well as smoking and may very well be one reason smoking has been associated with chronic low back pain.[48]

How does cigarette smoke affect the vertebral blood flow? One explanation previously discussed is the effect of nicotine on the sympathetic nervous system creating decreased peripheral blood flow and venoconstriction.[1,3,16,23,25,28-31] There have also been epidemiological studies from a variety of countries throughout the world that have clearly established a relationship between smoking cigarettes and the development and/or progression of the atherosclerotic process.[46] It has been suggested that nicotine may cause arterial endothelial cell damage, thus implicating this compound in the initial event related to atherogenesis.[46] Cigarette smoking is widely accepted to have a significant positive association with atherosclerosis in coronary, aortic, abdominal and peripheral arteries.[46]

Endothelial injury has been considered to be the absence of endothelium and presence of platelets.[46] At the simplest level, these changes could result in the vessel wall being more permeable to lipids or having reduced anti-platelet properties.[46] Smoking promotes platelet adhesion to the endothelium of the artery.[46] It has been established that platelets play an essential and pivotal role in the pathogenesis of the atherosclerotic and induced vascular occlusion diseases.[46]

Nicotine increases sympathetic nerve activity and releases catecholamine in adrenal glands.[46] This

generalized physiologic response could potentially influence the atherogenic process by several mechanisms: a) increase in circulating blood levels of free fatty acids, b) increase in plasma fibrinolytic activity, c) increase in platelet aggregation, d) an effect on some steps in the blood coagulation pathway, e) increased blood pressure, flow velocity and blood shear rate, and f) an increase in blood glucose levels and arterial basic metabolic rates.[46]

Vertebral blood flow is also affected by the increased blood viscosity in heavy smokers.[43] Blood viscosity has been found to be 50 percent higher in nonsmokers than in heavy smokers. This alteration in blood rheology leads to a malnutrition of the highly bradytrophic intervertebral disc.[43]

Blood flow may also be affected by nicotine-induced vasoconstriction. Data has shown that smoking reduces rectal blood flow.[31] Comparable work on the effect of smoking and nicotine on blood flow to other organs has shown constriction in cutaneous blood vessels with decreased blood flow to the hands and feet.[31] This decreased blood flow may be due to increased vascular tone.[31]

Laing and Grenhalgh[46] have found that over 97 percent of patients presenting with peripheral vascular disease are smokers. The association between cigarette smoking and the development of atherosclerosis, in particular peripheral vascular disease, is strong. However the mechanisms which

result in cigarettes being such potent risk factors are unclear.

Ross and Glomsett[46] have put forward the "response to injury" hypothesis, which links endothelial damage and smooth muscle proliferation to the incorporation of lipids and fibrin into the arterial wall. If this hypothesis is correct, a likely target on which cigarette smoke could exert its effect is on the endothelium.

Woolf[46] has shown that after a single brief exposure to cigarette smoke, endothelial cells became swollen and that longer exposure causes the appearance of sharply demarcated holes which may be the stomata of active pinocytic vesicles; however, the endothelium maintained its integrity. Cigarette smoke increased epithelial permeability.[46] It is possible that such a change in the permeability of the arterial wall would result in an influx of lipids and other plasma macromolecules into the arterial wall.[46]

Endothelial damage and platelet activation appear to be among the more important mechanisms involved in the pathogenesis of atherosclerosis.[46] Cigarette smoking has been associated with both endothelial damage and platelet aggregation. Endothelial injury also leads to vasospasm instead of vasodilation in response to products released by aggregating platelets.[46]

Substantial evidence has been elucidated regarding the mechanisms that link cigarette smoking with the development

and accelerated progression of atherosclerosis.[46] Influences on platelet function, lipid metabolism, coronary artery tone, and the sympathetic nervous system, the endocrine system, and oxygen transfer and dissociation from hemoglobin have been identified.[46]

The circulatory changes seen with cigarette smoking which could affect vertebral blood flow include decreased peripheral blood flow and vasoconstriction, decreased oxygen tension in discs, decreased fibrinolytic activity, increased blood viscosity and development and/or acceleration of atherosclerosis. Whenever vertebral blood flow is compromised, the disc may not receive adequate nutrition and then will be susceptible to injury.

Osteoporosis

Women smokers have been shown to have significantly lower spine bone mineral density and a tendency for lower bone mineral density at other sites.[9,18,35,36,56] Bone mineral content has also been found to be much less in elderly male heavy smokers.[36] There is an increased risk of microfractures of the trabeculae due to the osteoporosis which might be responsible for some low back pain.[9,10,34,57]

Osteoporosis results from a gradual decrease in the amount of bone present in our bodies resulting in an increased susceptibility to fracture.[37,58] Loss of bone mass occurs as a normal part of the aging process; however,

in the individual with osteoporosis, the loss is so extensive that the bone mass falls below the threshold of a fracture.[58] Changes in bone are attributable to the activity of groups of osteoclasts and osteoblasts.[58] Osteoclasts appear on the surface of bone and are responsible for resorption, which is the process whereby calcium phosphate crystals are removed from the bone and are absorbed by the blood.[58] After the resorption phase is completed, osteoblasts appear and are responsible for new bone formation.[58]

Risk factors of osteoporosis include: lack of regular exercise, early menopause, aged forty or older, female, white, cigarette smoking, family members with osteoporosis, underweight, heavy alcohol use, certain medications such as cortisone-like drugs and aluminum containing antacids, certain medical problems (rheumatoid arthritis, emphysema, chronic bronchitis, hyperthyroidism, some types of stomach surgeries, and diabetes mellitus), low calcium in diet, injuries and falls.[37,56]

Osteoporosis is very common in women after menopause.[37,58] It is thought that the lower level of estrogen after menopause is the basic problem and causes more rapid removal of the bone.[37] Estrogen has a protective effect on the bone by suppressing resorption.[58] Estrogen improves calcium absorption in the intestinal tract and decreases calcium losses in the urine.[58] With increased

levels of estrogen, the active form of vitamin D in the circulatory system is elevated, and the estrogen then stimulates production of calcitonin, which prevents removal of calcium from the bone.[58] As the amount of estrogen decreases at menopause, more bone is lost, less bone formed, and bones become thinner.[37]

The earliest fractures to occur after menopause, those of the distal radius and spine, have been ascribed to a rapid loss of trabecular bone due to the hormonal changes, mainly estrogenic.[58,59] A significant correlation exists between the bone mineral content and ultimate compressive strength of the lumber vertebrae.[57] The number of trabecular fracture calluses in vertebral bodies increase with decreased bone mineral content.[57]

In women, cigarette smoking is associated with earlier menopause and increased risk of osteoporosis, believed to be associated with lower levels of estrogen in smokers compared to nonsmokers.[9,10,16,18,33,34,37,56,60] Smokers have substantially and significantly lower levels of all three major estrogens (estrone, estradiol, estriol) in the luteal phase of the menstrual cycle.[34]

Researchers have postulated that reduced bone mineral density may result from lower body weight among smokers or a direct effect of smoking on estrogen metabolism.[18,60] Recent reports, however, have indicated no association between estrogen levels and body weight in premenopausal

women.[34] Low estrogen excretion of smokers could reflect lower estrogen production.[34] It is conceivable that through liver damage or other mechanisms, smoking may lead to changes in the metabolism of estrogen.[34] Recent evidence suggests that nicotine and other alkaloids in tobacco inhibit the formation of estrogen by inhibiting an aromatase enzyme in granulosa cells and placenta tissue.[16]

Several studies have found a significant negative effect of smoking on spine bone mineral density.[18] Daniell[56] found that post-menopausal smokers aged 60 to 69 years exhibited much more bone loss than did nonsmokers. He also observed that middle-aged men and women with symptomatic osteoporosis were almost exclusively heavy cigarette smokers.[56] Seventy-six percent of the patients with idiopathic osteoporosis and secondary osteoporosis were smokers, compared to 43 percent of office patients.[56] His study also found spontaneous menopause occurring in smokers on average of 5.3 years earlier. The earlier menopause of smokers could be expected to advance the development of osteoporosis by several years, thus increasing the risk of fractures.

Another study found bone mineral content to be much less in male heavy smokers aged 61-90 years.[36] This study found sensitivity to calcitonin of rats inhaling tobacco smoke was less than in control rats.[36] It appears that the tobacco smoke reduced the sensitivity to calcitonin.

Calcitonin protects bone tissue against resorptive effects, hence decreased sensitivity to it may offer an explanation for the osteoporogenic effect of smoking, especially because calcitonin resistance can be found in pathologic menopausal osteoporosis.[36]

Cigarette smoking is thought to double your risk of osteoporosis.[37] According to information in current literature, this is more than likely due to decreased estrogen production in smokers and also the effect of smoking on calcitonin resistance.[18,34,36,37,56]

Social and Psychological Traits

Finally, smoking has been associated with low back pain because of social and psychological traits. Smokers may have a psychological and social profile that both relates to the smoking habit and places them at a greater risk of symptomatic low back pain. Numerous studies have attempted to outline personality and social traits of smokers. Persons with chronic low back pain have also been studied extensively from a psychological and behavioral point of view, but there is no conclusive evidence as to whether smoking contributes to low back pain because of these traits in an etiological fashion or simply is a marker for these common psychological and social traits. Certainly, behavioral factors and the person's psychological adjustment

are increasingly being recognized for their role in determining the cause and outcome of low back pain.[61-65]

Several studies have shown strong correlations with certain psychological features and poor results from back surgeries.[62,63,65,70] Pooling of biological and psychosocial factors appears promising in prediction of surgical outcomes.[62] Wilfling et al.[69] found the relationship between current psychological status and success or failure of surgical fusion to be striking. The neurotic triad of hypochondriasis, depression and hysteria characterized the poor and fair outcome groups and was absent from the good outcome group.[69] The good group was better integrated emotionally.[69] Multiply operated patients had a high elevation on all three Minnesota Multiphasic Personality Inventory (MMPI) scales of hypochondriasis, depression and hysteria.[63,67,69]

Svensson et al.[9] found the feelings of worry and tension were associated with low back pain. There is some evidence of an association between low back pain and psychological factors such as anxiety, depression, hysteria and hostility.[20,64,71,72] Heliovaara et al.[42] found that symptoms which suggested psychological distress in women were a significant predictor for hospitalization due to herniated lumber disc or sciatica. The presence of distress symptoms crudely doubled the risk of herniated intervertebral discs or sciatica in women.[42]

Persons with low back pain who have had a history of divorce, separation, and widowhood were found to be lower achievers in a rehabilitation program than those who were married and had no such history.[20] Overall, there is a higher prevalence of back pain among no-longer married persons over age 35.[13,73] This may be due to the fact they must contend with decreased social support and increased responsibility in all areas of activities of daily living.[73] This may lead to physical and emotional stress, which in turn may precipitate or aggravate back pain.[73]

Emotional stress may be a factor in providing a periodic increase in pressure within the annulus.[72,74] Reaction to stress could lead to increase in size of the nucleus pulposus and ultimately lead to disc protrusion.[72,74]

Significant differences have been noted between back patients and controls regarding personality characteristics.[21] Back patients more frequently had a heavy physical job with workshifting and subordinate positions.[21] They also report more job dissatisfaction.[21,67] Back patients also demonstrated more sick listing under various other diagnoses and abuse of alcohol.[13,21,71,75]

Wilfling et al.[69] identified several factors in his study that may contribute to the problem of low back pain. These factors included: lower level of formal education,

unskilled or semi-skilled occupation, poor occupational stability, marital difficulties, and psychiatric contacts during the natural history of illness.

Studies have also shown differences in personality and social traits of smokers compared to nonsmokers. Smokers show more prevalence in changing jobs frequently, higher rate of divorce, lower academic achievement, drinking more, rebellion against authority, lower levels of physical activity, and difficulty sleeping.[1,3] Smokers also tend to show poorer adjustment and chronic anger.[2,40] Nelsen[40] reported evidence that smokers may be predisposed to emotional disturbance.

Cigarette smokers also tend to engage in other potentially deleterious health behaviors.[19] Lee and Markides[19] found positive associations between smoking status and heavy caffeine and alcohol consumption. Younger men who smoke have reported greater activity limitation due to poor health.[19]

Smoking may interact with other risk factors producing a synergistic increase in risk of negative health outcomes.[19,76] For example, this type of interaction with other risk factors like genetic predisposition, stress, personality, and drinking can influence the initiation of cancer or coronary heart disease.[76]

Jamison et al.[5] investigated the physical and psychological factors involved in the relationship between

cigarette smoking and chronic low back pain. Pain and smoking frequency are associated with worry, tension, fatigue, and perception of stress.[5] Smokers tended to present more maladaptive pain behaviors.[5] These include decreased activity, reliance on medications, and expressions of emotional distress.[5] Smoking is known to accelerate the metabolism of many drugs and the efficacy of sedatives and analgesics appeared to be reduced in cigarette smokers.[5] Chronic pain patients may be prone toward cigarette smoking because of its supposed relief from anxiety and possible relief from pain.[5]

It is possible that chronic smokers have a psychological profile that both relates to the smoking habit and places them at a greater risk for lower back pain. One hypothesis deals with the link of cigarette smoking to anxiety and depression.[8] Since these may exacerbate or prolong back pain, smoking may in part be a marker for these psychological traits.[8] Smoking may also be a marker for a complete combination of social traits that are associated with increased risk of low back pain.[8] This habit is most prevalent in lower socio-economic groups for whom physical job demands, life stresses, income, and other health habits may increase the risk of back pain.[8] Smoking is also associated with greater alcohol intake and possibly other dietary and lifestyle differences.[8] Several studies mentioned decreased physical activity among smokers.[19] It

has been shown that decreased physical activity is significantly related to the development of osteoporosis which may explain some problems with low back pain.[66,77]

As mentioned previously, stress is associated with low back patients as well as cigarette smoking. Stress is known to produce accompanying physiological alterations.[20] A common clinical example is chronic nervous tension which leads to prolonged hypertonicity of the muscles with associated stress on the osseous insertions producing a low grade periostitis, increased pressure on the nerves within the muscle group, and associated muscular fatigue. A second linkage arises in situations in which social and psychological factors may aggravate a functional or structural condition or facilitate the influence of physical or biological disease agents.[20]

Many similarities in personality and social traits have been reported in persons with low back pain and in cigarette smokers. How these traits can actually be etiological factors or risk indicators for low back pain has not been established, as has been evidenced by the many theories explained above.

CHAPTER V

CONCLUSION

Cigarette smoking is known to be detrimental to an individual's health in many ways. Much research has been done on nicotine to show both physiological and psychological effects of this drug. However, even with the extensive information concerning cigarette smoking and nicotine, its direct effects on low back pain have not been firmly established.

In the last ten years, various authors have studied the link between cigarette smoking and low back pain. The four theories explored in this paper were the effects of the chronic cough present in cigarette smoking, the numerous vascular changes that occur in the presence of nicotine, the acceleration of osteoporosis in cigarette smoking, and the smokers social and psychological traits which increases their risk of back pain.

In the opinion of this author, the best developed theory is that of vascular changes associated with nicotine. Nicotine is associated with decreased peripheral blood flow, defects in fibrinolysis, and the atherosclerotic process.[16,23,25,28-31,35,45-48] Because the central segments of the disc depend on diffusion for their

nutrition, it places the disc in a precarious balance between nutrient utilization and blood supply.[49-51] If the end-plate is partially blocked or blood flow is less, adverse effects on disc metabolism occur, making the disc more susceptible to injury.[7,8,10,12]

The other theories presented in this paper also present some probable links between cigarette smoking and low back pain. Cigarette smoking may just be one of many risk factors of low back pain, or it may be a causative factor. Clearly, more research must be done in this area to ascertain the exact role of cigarette smoking and nicotine in the onset and continuation of low back pain.

Even though conclusive evidence does not exist explaining the link of cigarette smoking and low back pain, existing studies have shown a strong correlation. This correlation would seem to suggest that healthcare providers should not overlook this factor when treating low back pain patients. Whether or not a patient continues to smoke may have a significant bearing on the outcome of the treatment and rehabilitation success of patients with low back pain.

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